working no. 7

134

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 36 Number 1

January 15, 1952

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
Volume 36 — January 15, 1952 — Number 1

IN THIS ISSUE

Results of a test, designed to evaluate losses in cotton caused by angular leaf spot infection, are reported by L. S. Bird, page 3.

Dewey J. Raski reports on the host range of the sugar-beet nematode in California, page 5.

The Ascochyta leaf spot in tobacco plant beds in South Carolina is tentatively identified as A. gossypii, according to Q. L. Holdeman and T. W. Graham, page 8.

Alice L. Robert and William R. Findley, Jr. report diseased corn leaves as a source of infection in artificial and natural epidemics of Helminthosporium turcicum, page 9.

Yellow spot of winter wheat has been observed causing severe reductions in yield for the first time in Michigan, according to Ed Andrews and Wm. Klomparens, page 10.

D. E. Ellis reports Ascochyta leaf spot of bean in eastern North Carolina this year, page 12.

Charles Drechsler reports bean stem rot caused by several Pythium species in Delaware and Maryland, page 13.

In South Carolina the increased susceptibility of the Palmetto cucumber to downy mildew, presumably due to a new race of the pathogen, is reported by W. M. Epps and W. C. Barnes, page 14.

According to W. G. Keyworth in a special study, tests showed that strains of Verticillium albo-atrum pathogenic to potatoes were present in all reported cases of premature wilting in potato fields in Connecticut, page 16.

D. L. Gill reports that the results of tests show that tung-nut meal is of no value as a soil treatment for the control of root-knot nematodes. Parathion mixed in the soil was of value in root-knot nematode control, page 18.

J. H. Graham reports on new wildfire symptoms on soybean and the preservation of three bacterial pathogens of soybean in culture, page 22. Of the three methods tried storage under mineral oil was the most successful.

J. C. Carter reported that during 1951 eleven additional trees infected with Dutch elm disease were found in four counties in Illinois, page 24.

The distribution and spread of oak wilt in Illinois is reported by J. C. Carter, page 26.

L. O. Weaver and W. F. Jeffers report that as a result of a survey of oak wilt in western Maryland only a small number of trees had been killed within four years and there was no evidence of rapid spread of the disease, page 28.

In New York, Louis G. Nickell reports a species of Ganoderma probably pathogenic to Sassafras, page 28.

Unusual occurrences of plant diseases, page 30, include the reappearance of silver leaf in the apple orchards of Washington, by Roderick Sprague and H. H. V. Hord; and Phytophthora cinnamomi on avocado in Mexico and Costa Rica, and other avocado diseases in Mexico, by George A. Zentmyer, page 31.

Some corrections, page 31.

November weather, page 32.

889206

THE IMPORTANCE OF RESISTANCE TO THE BACTERIAL BLIGHT (ANGULAR LEAF SPOT) DISEASE OF COTTON

L. S. Bird[1]

Bacterial blight (Xanthomonas malvacearum) of cotton occurs wherever the crop is grown. In this country it is a major disease in the Southwestern production areas and in Texas is especially severe in the Blackland and High Plains areas.

The nature of the disease makes it difficult to obtain accurate estimates of yield losses under commercial production conditions. For this reason, a test designed to evaluate losses caused by bacterial blight infection was conducted during 1951. This paper reports the results of the test which was conducted on the Main Station Farm at College Station, Texas.

MATERIALS AND METHODS

The test was a randomized split-plot design with 12 replications. Four strains were entered. They were: Stoneville 20, a highly resistant strain having only a fair yielding ability; Deltapine, a susceptible commercial variety having high tolerance to the disease; Stoneville 2B, a susceptible commercial variety having medium tolerance; and Acala, a susceptible commercial variety having low tolerance. Stoneville 20 is not a commercial type. It is used as a source of resistance in breeding programs to develop blight resistant varieties of cotton. Therefore, normally its yield will not compare favorably with the commercial strains used.

The plot length for each strain in a replication was 50 feet. Half of each plot was inoculated (spraying a bacterial suspension through open stomata, on the lower side of leaves) with the blight causal organism and the remaining half was not inoculated. The two halves were designated blight-infected and blight-free. The blight-infected plots were inoculated twice. The first inoculation was made at about the fourth true leaf stage and the second during the first part of the flowering period.

Bacterial blight is transmitted very easily by wind-driven and splashing rain. Therefore, the experiment was designed to decrease as much as possible the natural spread of the disease to the blight-free plots. This was done by: planting alternate rows to highly resistant material; spacing replications 5 feet apart and planting this space to resistant material; leaving 2 feet between the blight infected and blight free plots within replications and planting this space to resistant material. The test plot did not adjoin cotton on any side, two sides were bordered by corn, one side by sorghum and the fourth by an uncultivated field. Yield data and lint percent were obtained for each plot and the data were studied by analysis of variance methods.

RESULTS

The 1951 growing season was very dry. The average yield was low and no natural blight infection occurred, this allowing the uninoculated plots to remain blight-free. Also, since no natural infection occurred, there was no boll infection.

As a result of the inoculations, a medium to heavy degree of leaf infection occurred in the infected plots of the susceptible strains. No infection developed in the inoculated plots of Stoneville 20. The infection caused a pronounced amount of leaf shedding in susceptible strains that did not occur in the blight-free plots. There was an observably larger number of leaves in the blight-free plots as compared with the remaining leaves in the infected plots.

The analysis of the lint percent data showed no effect of bacterial blight infection on that factor. The components of the analysis of variance for the yield data are given in Table 1.

As expected the F values for replications and strains are highly significant. The highly significant F value for treatments indicates that the yields of the blight infected and blight free plots were different. Previous work showed that the four strains in the test differed in their reaction to blight infection (Stoneville 20 - highly resistant, Deltapine - highly tolerant, Stoneville 2B - medium tolerance, and Acala - low tolerance); therefore the treatments x strains interaction was expected to be significant. An examination of the yield data in Table 2 also suggest that it should be significant. An explanation for the non-significant treatment x strains interaction is not evident.

The average yield per acre for the blight-infected and blight-free plots are given in Table 2. This table shows the significant decrease in yield of seed cotton for the susceptible strains while there was no decrease for the highly resistant Stoneville 20. The amount of loss varied, with the

[1]Instructor, Dept. Plant Physiology and Pathology, Texas Agricultural Experiment Station, College Station, Texas.

Table 1. Analysis of variance of yield per plot for the four strains of cotton that were blight-infected and blight-free.[a]

Variation due to	Degrees of freedom	Mean square	F value
Reps.	11	13,932	5.13[b]
Strains	3	32,073	11.81[b]
Error a	33	2,717	
Treat.	1	16,276	14.25[b]
Treat. x strains	3	2,346	2.06
Error b	44	1,142	
Total	95		

[a]Yield weight in grams.
[b]Exceeds the 1 percent point.

Table 2. Mean yield per acre for four strains of cotton that were blight infected and blight free.

Strains	Yield, pounds of seed cotton per acre[a]		Decreased yield due to blight infection[b]	Percent loss due to blight infection	Monetary loss per acre[c]
	Blight free	Blight infected			
Stoneville 20 (highly resistant)	213	214	-1	----	----
Stoneville 2B (Susc. with tolerance)	266	235	31	11.7	$3.88
Deltapine (Susc. with tolerance)	253	216	37	14.6	$4.63
Acala (Susc. with low tolerance)	180	127	53	29.4	$6.63
Averages				18.6	$5.05

[a]Average yield for test was 213 pounds of seed cotton per acre.
[b]Difference required for significance: odds 19:1, 16.0 pounds; odds 99:1, 21.4 pounds.
[c]Based on 12.5¢ per pound for seed cotton.

tolerant strains (Stoneville 2B and Deltapine) having less loss than the low-tolerant Acala strain. Just why Deltapine, which has a higher level of tolerance than Stoneville 2B, had a greater loss can not be explained at this time.

As pointed out, no boll infection developed in the test. Therefore, the reductions in yield are attributed to the leaf phase of the disease. The average loss of 18.6 percent seed cotton yields, or $5.05 monetary loss per acre, for the blight susceptible strains as compared with no loss for Stoneville 20, emphasizes the importance of blight-resistant cotton.

SUMMARY

A test designed to evaluate losses in cotton yields caused by bacterial blight infection showed that a highly significant reduction in yield of seed cotton was caused by the disease.

Susceptible strains had an average loss of 18.6 percent caused by the disease, while Stoneville 20, a highly resistant strain, had no loss. The average monetary loss per acre for the susceptible strains was $5.05.

TEXAS AGRICULTURAL EXPERIMENT STATION, COLLEGE STATION COOPERATING WITH THE DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, U. S. DEPARTMENT OF AGRICULTURE

ON THE HOST RANGE OF THE SUGAR-BEET NEMATODE IN CALIFORNIA

Dewey J. Raski

The host range of the sugar-beet nematode, Heterodera schactii Schmidt, as reported in the literature, includes a great many species of plants in as many as 25 families. The range was even greater until Franklin (1) established the specific status of H. avenae, H. göttingiana and H. rostochiensis. Since that time H. schachtii has generally been considered as capable of developing on some members of the Chenopodiaceae and Cruciferae and occurring sporadically on a number of other hosts, principally weeds.

The practical importance of establishing the true host range of H. schachtii is self-evident. The entire theory of crop rotation as a control measure depends on an accurate understanding of the host plants suitable to this nematode. From a taxonomic standpoint there has been increasing emphasis on the host range as an important guide toward the identification of closely related species of Heterodera even when there is considerable overlapping of ranges.

One important difficulty encountered in attempting to determine the host range of any species of Heterodera, in the absence of adequate morphological differences, is the possibility of mixed populations in field soils. Jones (2) reports many new hosts for sugar-beet nematode in field tests. The consistency of his findings greatly reduces the likelihood of identical mixtures occurring in several different localities. However, field tests have always been accepted with some reservations unless substantiated by additional tests using nematodes found on the roots of the test plants. The re-inoculation of sugar-beets as well as the original host by progeny selected from the roots of the new host would be valuable in more definitely establishing the species involved.

Host range tests of H. schachtii have been conducted in this laboratory from 1947 to the present time using soil collected from a sugar-beet field near Salinas, California. In one of the first tests, seedlings of 20 plants were transplanted to pots of the field soil and allowed to grow for seven days. The plants were then washed free and the roots stained with acid fuchsin in lactophenol. Larvae were found in the roots of black mustard, spinach, cucumber, carrot, Golden Queen tomato, egg plant and onion. The invasion of egg plant and onion could not be duplicated in later tests. Negative results were obtained for leek, red clover, alfalfa, black-eyed beans, Atlas barley, lettuce, wheat, oats, cotton, salsify, celery, parsley and parsnip.

The test was repeated for Golden Queen tomato, carrot and cucumber. The plants were grown in the infested soil for 37 days in this case. The stained roots of the tomato plants contained three females (three eggs present in one of them), three males and eleven young in various stages of development. The carrot and cucumber roots held 36 and 31 larvae, respectively. None of these larvae was developed beyond the second stage. Several tests confirmed this development on Golden Queen tomato as well as on Jubilee (both yellow varieties) but not on Earliana, a common red variety.

Five cysts taken from the roots of Golden Queen tomato were transferred to sterilized soil and planted with seeds of Golden Queen tomato. After 38 days only one third-stage male was found in the roots.

Further host range studies were undertaken when sweet pea, Lathyrus odoratus, was found to be a host of H. schachtii. Additional plants were then tested in the same field soil and the results are indicated in Table 1.

Females and cysts were taken from the roots of several of the above plants and transferred to pots of sterile soil. Seeds of the original plant or of sugar beets were then planted in the soil with the results indicated in Table 2.

Sesbania macrocarpa was also tested by planting seeds in three separate pots of the same field soil. The first two pots, examined after 50 and 75 days, respectively, showed negative results. The roots of the seedlings in the third pot grown for 96 days held 30 ♀♀, 25 ♂♂, and many larvae in various stages of development on the roots as well as many more males washed from the soil. Twenty females collected from the roots of the Sesbania seedlings were transferred ten each to two pots of sterile soil and planted with seeds of Sesbania and sugar beets, respectively. After 70 days growth no nematodes could be found on the roots of the sugar beets. One fourth-stage female was found on the roots of the Sesbania and one male washed free from the soil.

Representative specimens were preserved for permanent mounts whenever possible during all these tests. None of the specimens showed any differentiation morphologically from specimens taken on sugar beets grown in the same field soil.

Table 1. Results of additional plants tested.

Host Plant	Nematodes on roots after 75 days.
Sugar beet	Heavily attacked; all stages present.
Golden Queen tomato	10 ♀♀, 5 cysts; 1 ♂; many larvae in various stages of development.
Annual lupine	30 ♀♀; 9 ♂♂; 16 larvae in various stages.
Golden Wax bush bean	5 ♀♀; 5 cysts; 28 developing ♂♂.
Iron cowpea	9 ♀♀; 13 cysts; many larvae in various stages.
Garden pea	12 ♀♀; several developing larvae.
Purple vetch	Several males and developing larvae.
Sweet clover	3 second-stage larvae, 1 third-stage larva all from one small locality only.
Crotalaria spectabilis	Many undeveloped larvae.
Castor bean	One undeveloped larva.
Sweet pea*	4 ♀♀ and many developing larvae in all stages.
Nightshade*	3 ♀♀

*The nightshade was a contaminating weed in the pot in which sweet pea was grown and males which were washed from the soil could not be assigned to either host with certainty.

Table 2. Results of seeds planted of original plant or of sugar beets.

Original Host Plant	Subsequent Host Plant Grown for 71 Days	Nematodes Recovered on 2nd Host
5 ♀♀ from lupine to	lupine	0
5 ♀♀ from lupine to	sugar beet	8 ♀♀, 7 ♂♂
5 cysts from Iron cowpea to	Iron cowpea	0
3 cysts from Iron cowpea to	sugar beet	0
4 cysts from Golden Wax bush bean to	Golden Wax bush bean	0
1 cyst from Golden Wax bush bean to	sugar beet	2 ♀♀
2 ♀♀, 2 cysts from Golden Queen tomato to	Golden Queen tomato	Roots rotted by fungus
2 ♀♀, 2 cysts from Golden Queen tomato to	sugar beet	5 ♀♀, 6 ♂♂, 2 fourth-stage larvae
2 ♀♀ from nightshade to	sugar beet	0

Several collections have also been made in sugar-beet fields in various parts of California from the roots of Amaranthus retroflexus, A. graecizans, Chenopodium murale and Solanum nigrum. The collections consist of mature female nematodes taken directly from the roots of the

various plants and no morphological differences have been found to distinguish them from H. schachtii. In most of these cases the female nematodes were occasional to rare and only found after the examination of many plants.

DISCUSSION

Increasing evidence is being obtained which indicates that a true understanding of the host plant relationships of the sugar-beet nematode has still not been demonstrated. The information presented here shows the full range of plants suitable to this nematode has not been worked out. It has been known for a long time that there are some plants which can be invaded by the larvae of this nematode but are not suitable for development and growth of the larvae after penetration. Also, most of the plants which are readily attacked by the larvae in large numbers and are suitable hosts have been extensively tested. However, there are many plants which fall between these two categories. In the case of Sesbania macrocarpa the plants are readily attacked but progeny produced on these plants seem unable to develop on sugar beets or Sesbania. In other cases the plants are attacked by relatively few larvae which are not able to build up high populations on the plants. It is also possible that variations in host ranges between populations from different localities have not been conclusively ruled out.

The work described here is by no means conclusive but serves rather to point to the need for more fundamental work on the host plant relationships of H. schachtii as well as other species of Heterodera. Until our knowledge concerning these nematodes is more complete, it seems advisable to proceed somewhat more slowly with the assignment of specific status to populations showing host range differences unless there also exist adequate morphological differences which can be demonstrated.

SUMMARY

Host range studies of the sugar-beet nematode in California have been conducted in the greenhouse using field soil collected near Salinas, California. The larvae of this nematode were found to be capable of invading but not able to develop in the roots of carrot, cucumber and Crotalaria spectabilis. A number of plants not previously reported to be hosts were found suitable for reproduction, including two yellow varieties of tomato (Golden Queen and Jubilee), annual lupine, Golden Wax bush bean, Iron cowpea, Garden pea, sweet pea (Lathyrus odoratus) and Purple vetch. Females and/or cysts taken from roots of lupine, Golden Wax bush bean, and Golden Queen tomato reinfected sugar beets but were not able to attack the original host. Sesbania macrocarpa was also readily attacked by this nematode but reinfection of Sesbania or sugar beet could not be demonstrated with females from roots of Sesbania. Females of the sugar-beet nematode were also collected from the roots of Amaranthus retroflexus, A. graecizans, Chenopodium murale and Solanum nigrum found growing in various sugar-beet fields.

Literature Cited

1. Franklin, Mary T. On the specific status of the so-called biological strains of Heterodera schachtii Schmidt. Jour. Helmin. 18: 193-208. 1940.
2. Jones, F. G. W. Observations on the beet eelworm and other cyst-forming species of Heterodera, Ann. App. Biol. 37: 407-440. 1950.

DIVISION OF ENTOMOLOGY AND PARASITOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

ASCOCHYTA LEAF SPOT IN TOBACCO PLANT BEDS IN SOUTH CAROLINA

Q. L. Holdeman and T. W. Graham

In April 1951, a tobacco leaf spot was found at two locations in plant beds, from which an Ascochyta was isolated. At one location near Latta, South Carolina a single infected leaf was found causing a circular area of watery soft rot 25 mm. in diameter. At another location near Olanta, South Carolina the leaf spots resembled "frogeye," and as many as 18 spots were found on a single leaf when plants were about one-half transplanting size. Spots were up to 15 mm. in diameter, circular to irregular, and sometimes coalescing. Necrotic tissue was dry and occasionally broke away leaving a hole in the leaf. Ostiolate, dark, partially submerged pycnidia were present on the upper surface of diseased tissue.

The fungus was isolated, and the disease was reproduced in inoculation trials with both collections. Beginning as watersoaked spots, lesions were evident within 48 hours on young tobacco seedlings.

For purposes of comparative pathogenicity tests an Ascochyta isolate from cotton was obtained from Dr. C. H. Arndt. Both tobacco and cotton isolates showed a similar degree of pathogenicity when cotyledons of cotton seedlings and leaves of tobacco were inoculated. Sporulation of the three isolates was observed on both the inoculated hosts. Colonies of the fungus reisolated from both hosts were comparable in growth habit to the original isolates.

On field material collected at Latta pycnidia measured 22.0 - 47.1 microns; spores were 1 to 2-celled, hyaline, ovate to elliptical to irregularly oblong; occasionally constricted at the cross walls, and measured 2.7 - 5.4 x 6.0 - 10.8 microns. On material collected at Olanta pycnidia measured 33.7 - 53.9 microns; spores measured 3.3 - 5.4 x 8.1 - 12.1 microns. Comparison was made between cotton and tobacco isolates sporulating on inoculated tobacco seedlings. The size of the pycnidia and the spores fell within the same general range. The pycnidia were about half the size recorded by Elliot on cotton in Arkansas (1), but the spore measurements were within a comparable range. The proportion of length to width and the frequency of constriction at the median septa may be altered by the amount of moisture present when the spores are formed.

There are infrequent reports in the literature on the occurrence of Ascochyta nicotianae Pass. on tobacco. The fungus was described in Italy more than 75 years ago and was designated as a new species. No measurements were given in this description (6). In his book on tobacco diseases, Wolf (7) stated the fungus had been reported in several European countries and in Puerto Rico. Ascochyta on tobacco has also been reported from Formosa (5) and from France (3), where it was isolated from stem lesions. In the United States it was reported in Tennessee (4) and Washington (2).

It is almost impossible to make a definite determination based solely on the written description (6) of Ascochyta nicotianae. From pathogenicity tests on cotton and tobacco it is concluded that the cotton and the tobacco isolates used in this study belong to the same species. The Ascochyta collected on tobacco in South Carolina is tentatively identified as Ascochyta gossypii Sydow.

Literature Cited

1. Elliot, J. A. A new Ascochyta disease of cotton. Arkansas Agric. Expt. Sta. Tech. Bul. 178. 18 pp. 1922.
2. Fields, W. S. Summary of the more important plant diseases taken in connection with the insect and plant disease survey in the general vicinity of ports of entry from January 1945 to June 30, 1945. Plant Dis. Reptr. 29: 693-697. 1945.
3. Fourmont, R. Un Ascochyta parasite due tabac. Rev. Path. Veg. 25: 119-134. (Rev. Appl. Mycol. 17: 632) 1938.
4. Jehle, R. A. Diseases of field and vegetable crops in the United States in 1925. Plant Dis. Reptr. Suppl. 45: 127. 1926.
5. Matsumoto, T. Tobacco diseases in Formosa. Nation. Taiwan Univ. Fac. Agric. Mem. 1: 1-25. (Biological Abstracts vol. 22, entry 4940). 1946.
6. Saccardo, P. A. Sylloge Fungorum 3: 401. 1884.
7. Wolf, F. A. Tobacco diseases and decays. 454 pp. Duke Univ. Press, Durham, N. C. 1935.

PEE DEE EXPERIMENT STATION, OF THE CLEMSON AGRICULTURAL COLLEGE, FLORENCE, SOUTH CAROLINA

DISEASED CORN LEAVES AS A SOURCE OF INFECTION IN ARTIFICIAL AND NATURAL EPIDEMICS OF HELMINTHOSPORIUM TURCICUM

Alice L. Robert and William R. Findley, Jr.[1]

Northern leaf blight of corn caused by the fungus Helminthosporium turcicum Pass. has become a disease of particular concern in many of the corn growing areas. Seed producers and corn breeders are well aware of the need for testing their inbred lines and making selections for resistance to the disease. As natural infections occur sporadically, it is advantageous to induce epidemics at will to facilitate selection for resistance. Since 1943 this has been done at the Plant Industry Station by using quantities of inoculum made from pure cultures of the fungus. This has involved considerable laboratory work and time.

A simple way of initating and spreading an epidemic was tried on a small scale at the Plant Industry Station in 1951. This was done without laboratory culture work and with a minimum of time. It might be of help to those whose facilities and time are limited.

In October 1950, leaves from corn plants in the field that were heavily infected with Helminthosporium turcicum were collected and placed in the corn seed dryer at about 40°C for 4 to 7 days or until they were thoroughly dry. They were stored in an attic of uncontrolled temperatures until the spring of 1951. Tests were made immediately after drying and again in the spring to determine if the fungus in the lesions was still alive. Spores on the surface of the leaves were dead and did not germinate after drying but when leaves with lesions were put in a moist chamber at 15°C. and room temperature, conidia were formed within 1 to 4 days. Conidia also were formed when the test was made again in the spring.

Inoculations in the field were made on a susceptible corn hybrid using the dried infected leaves. Two methods were followed on each of two separate set of rows. (1) The leaves were ground in a Wiley mill and screened through a 40-mesh screen. This was mixed with water about 125 cc. per gallon and put into the leaf whorl of young corn plants in the same manner as inoculations with pure cultures[2]. Eight such inoculations were made, two each week for four weeks. (2) Fairly large pieces of the dried leaves were scattered variously between the rows and around the plants. At least one piece was close to each plant. This inoculation was made only once, 25 days after planting.

Both methods resulted in infection. In the first, lesions were seen for the first time 10 days after inoculation, scattered singly on a few plants. Secondary spread began after 31 days, as soon as it was initiated from the inoculations with pure cultures. Infection was not well established by the time plants tasseled. First lesions from the second method were seen 9 days after inoculation and they were plentiful in 11 days. There was an abundance of lesions on the lower leaves after 31 days and a fairly heavy infection of the plants had developed by tasselling time.

Generally, it takes from 7 to 10 days after inoculation for lesions to appear. Within 1 or 2 days after fully developed lesions are formed, conidia are produced that are transferred to other leaves causing the secondary spread lesions. It takes from 7 to 10 days for these to become apparent. After that new lesions develop progressively fast, if environmental conditions are suitable. With the first method used the spread must begin from initial leaf lesions caused by the inoculum. With the second method, conidia form on the old leaves within 1 to 4 days, if conditions are favorable, and continuously are formed and contribute to the spread of infection. Spread lesions show up in 7 to 10 days after conidia are formed. This advances the epidemic by about 7 to 10 days.

It is known that temperature and moisture greatly influence the spread of northern corn leaf blight and without the required environmental conditions spread would not be rapid. This would be true whether pure culture inoculum or diseased leaves were used.

These tests show that the mycelium of Helminthosporium turcicum will live over in the corn leaf tissue for at least one year after being dried out, although the spores do not remain alive. From this mycelium, conidia are produced under favorable environmental conditions which can infect young corn plants in the field. The use of diseased leaf material for inoculation in producing artificial epidemics not only saves laboratory work but under favorable conditions it may also advance the development of the disease by 7 to 10 days. This is important because it may enable the breeder to more effectively select resistant progenies before pollinating, thereby appreciably reducing the number of pollinations needed.

---

[1]Associate Pathologist and Assistant Agronomist, respectively.

[2]Elliott, Charlotte and Jenkins, Merle T. Helminthosporium turcicum leaf blight of corn. Phytopathology 36:660-666. 1946.

The fact that the fungus lives over a period in the leaf tissue strengthens the belief that natural infections are started from diseased refuse in corn fields. The effect of natural outside environment on the life of the fungus in leaf tissue is not known.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND

YELLOW SPOT OF WINTER WHEAT IN MICHIGAN IN 1951

Ed Andrews and Wm. Klomparens

Yellow spot of winter wheat caused by Helminthosporium tritici-vulgaris Nisikado has been observed causing severe reductions in yield for the first time in Michigan.

SYMPTOMS: Heavy infection (Fig. 1) was present at about the three-leaf stage of a field of Yorkwin wheat in Gratiot County. Spots were predominantly yellow early in the season, later becoming dark in the center. Yellowish tissue continued as a halo producing a roughly fusiform spot (Fig. 2). With age, the yellow coloration faded to a brownish-straw color. Sporulation occurred generally on the spots of the withered leaves.

FIELD HISTORY: The field was planted to wheat the second consecutive year with treated seed two years removed from certification. The county agent checked the commercial fertilizer application and found it adequate in analysis and amount per acre for a satisfactory yield of wheat. Pycnidia of Septoria tritici Rob. were found on leaves that overwintered, but new infections failed to develop in 1951. Some leaf rust occurred late in the season on leaf surface not occuppied by yellow spot lesions.

LOSSES: The yield from this field was 17 bushels per acre as compared with 35 bushels harvested in 1950. In view of the above history of the field, the loss was attributed primarily to yellow spot.

In another field in the same county on which wheat was planted four consecutive years, the leaves were almost completely covered with yellow spot lesions by the soft dough stage. At harvest time all heads sampled yielded poor quality, shrivelled grain. Lighter infections of the disease were observed in Kalkaska, Isabella and Monroe Counties during 1951.

Late in the season of 1950, H. tritici-vulgaris was collected on wheat in the vicinity of East Lansing, Michigan, but it was not possible to determine the extent of injury for that year.

FUNGUS MORPHOLOGY: Measurements of 80 conidia scraped from the lesions range in size from 14 x 30 microns to 24 x 156 microns. They average 18.8 x 110.6 microns.

Fifty conidiophores vary from 6.1 to 10.5 microns in width and from 26 to 360 microns in length. The average diameter is 8.8 microns.

The enlarged basal cell width varied from 10 to 18 microns.

The measurements of these structures are slightly larger than those listed by Sprague (2).

Other distinguishing spore characteristics including color, method of germination, number of septations, possible constriction of the septum, and curving to one side were found (Figs. 4 and 5). The characteristic light color toward the apex of the conidiophore (Fig. 3) as reported by other workers was found (1, 2).

Literature Cited

1. Raabe, A. Helminthosporium tritici-vulgaris Nisikado, Erreger einer Blattfleckenkrankheit des Weizens. Phytopath. Ztschr. 10: 111-112. (Rev. Appl. Mycol. 16: 664) 1937.
2. Sprague, Roderick. Diseases of Cereals and Grasses in North America. The Ronald Press Company, New York. 1950.

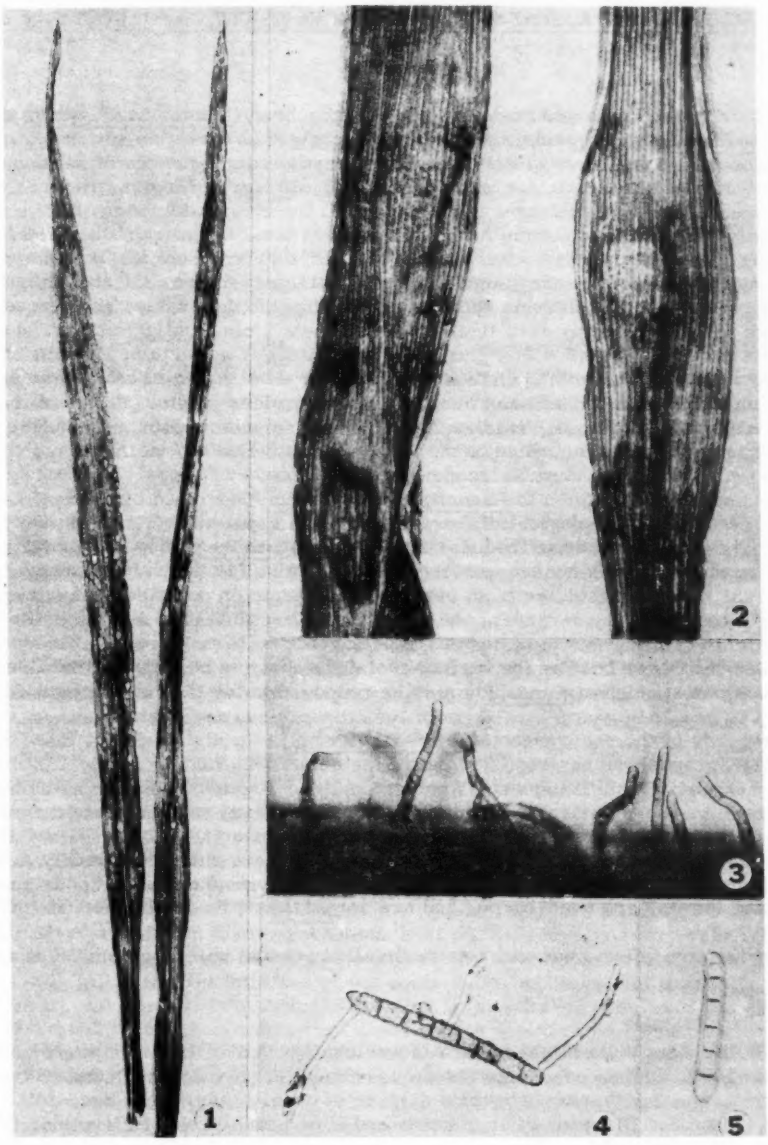


FIGURE 1. Yorkwin leaves infected with H. tritici-vulgaris causing yellow spot.
FIGURE 2. Typical mid-season lesions caused by H. tritici-vulgaris on Yorkwin wheat.
FIGURE 3. H. tritici-vulgaris conidiophores arising predominantly as single units, and showing progressive failure of color formation toward the apex.
FIGURE 4 and 5. Conidia of H. tritici-vulgaris showing germination from intermediate as well as polar cells, occasional curving to one side and constriction at the septa, and rounded, conical basal cell. Photography by Philip G. Coleman

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, MICHIGAN STATE COLLEGE, EAST LANSING

ASCOCHYTA LEAF SPOT OF BEAN IN NORTH CAROLINA

D. E. Ellis

A leaf spot disease of pole and bush varieties of snap bean (Phaseolus vulgaris) and lima bean (Phaseolus lunatus) with which a species of Ascochyta was consistently associated has been repeatedly observed and collected in the commercial bean producing areas of western North Carolina since 1948. The disease was also found in eastern North Carolina (Duplin County) in June, 1951. Greenhouse inoculations with monoconidial isolates of the fungus have established the causal relationship of the organism to the disease. In these tests it produced leaf and stem lesions on young plants in the cotyledonary leaf stage and also on plants with trifoliate leaves.

Typical young leaf spots are irregularly circular with gray to brown centers surrounded by a border of light green to yellow tissue. Older spots are light to dark brown and frequently zonate. The spots range in size from tiny dark flecks to large spots 2 cm. in diameter. The narrow dark brown to black stem and petiole lesions thus far observed reach a length of 1.8 cm. Lesions are rather common on the pulvini which result in considerable defoliation. The disease appears to be of minor importance on bush varieties, affecting primarily older plants. However, on pole varieties, especially pole snap beans, it is apparently more serious causing as much as 50 percent defoliation. Under conditions favorable to the fungus (relatively cool, wet weather) it may cause enough defoliation to be of considerable economic importance.

The fungus appears to conform to Ascochyta phaseolorum described by Saccardo in 1884 (4). Its spore size and other morphological characteristics also apparently fall within the range of those of Ascochyta abelmoschi described on okra (Hibiscus esculentus) by Harter (2) in 1918 and which occurs commonly on this host in western North Carolina (1). Results of cross-inoculation tests made in 1951 show that isolates from okra are pathogenic on pole and bush varieties of snap bean (Kentucky Wonder and Tendergreen) and lima bean (King of Garden and Henderson Bush) and that isolates from the various types of beans are pathogenic on okra (Louisiana Green Velvet). Symptoms produced on each host by the various isolates appear to be indistinguishable and the results suggest that one species of Ascochyta may be responsible for the disease both of beans and okra.

The only records of the occurrence of A. phaseolorum in the United States found in the literature are those by Sprague who has reported it on lima bean from Takoma Park, District of Columbia (7), and on snap bean from Issaquash, Washington, (9). Another species, A. boltshauseri (5), distinguished from A. phaseolorum by its much larger, frequently many septate conidia, has been reported on snap bean from Oregon (8) and on lima bean from North Carolina (3) and Pennsylvania (10). Both A. boltshauseri and A. phaseolorum apparently occur rather commonly in Europe. In Holland, according to Sneep (6), both species cause similar symptoms, affect pods and seeds as well as stems and leaves, are seed-borne, and are very destructive during periods of cool, wet weather.

Further studies are being made and a more detailed account will be published at a later date.

Literature Cited

1. Ellis, D. E. Ascochyta blight of okra in western North Carolina. Phytopath. 40: 1056-1058. 1950.
2. Harter, L. L. A hitherto unreported disease of okra. Jour. Agr. Res. 14: 207-212. 1918.
3. Prince, Alton E. Diseases of snap beans and lima beans in North Carolina. Plant Dis. Reptr. 28: 1015-1016. 1944.
4. Saccardo, P. A. Sylloge Fungorum 3: 398. 1884.
5. Saccardo, P. A. Sylloge Fungorum 10: 303. 1892.
6. Sneep, J. De Ascochyta-vlekkenziekte van de Boon (Phaseolus). Tikdschr. Pl Ziekt. 51: 1-16. 1945. (Abstr. in Rev. Appl. Mycol. 26: 276. 1947).
7. Sprague, Roderick. Host range and life history studies on some leguminous Ascochytae. Phytopath. 19: 917-932. 1929.
8. _____. Ascochyta boltshauseri on beans in Oregon. Phytopath. 25: 416-420. 1935.
9. _____. Ascochyta leafspot on snapbean in Washington. Plant Dis. Reptr. 32: 396. 1948.
10. Weiss, Freeman. Check list revision. Plant Dis. Reptr. 30: 66-74. 1946.

NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION, RALEIGH

BEAN STEM ROT IN MARYLAND AND DELAWARE CAUSED BY SEVERAL PYTHIUM SPECIES

Charles Drechsler

Pythium myriotylum Drechsl. is a fungus requiring high temperatures for its best development. Along the Atlantic seaboard it is known to occur as a plant pathogen in Florida, Georgia, and South Carolina. In Florida, certainly it is widely active during the long hot season, causing damping-off, root-rot, stem rot, and fruit rot of various crop plants. In our northern States it has come to light occasionally as a cause of cottony rot in vegetable products of southern origin. I have not recognized it among the pure cultures isolated by me since 1922 from diseased plants found growing outdoors in the District of Columbia and in adjacent portions of Maryland and Virginia. During the last 18 years, it must be admitted, my efforts in isolating root-rotting fungi have in conformity with rules governing the transportation of cultures been restricted very largely to materials originating in a relatively small area.

Six years ago Pythium myriotylum appeared unexpectedly in a greenhouse at the Plant Industry Station, near Beltsville, Maryland, as the pathogenic agent in more than a dozen bean (Phaseolus vulgaris L.) seedlings which on June 25, 1946, were found falling over, though they were then twelve days old and about 15 cm. high. In all the affected seedlings a basal portion of the stem, extending 2 to 5 mm. above the soil line, was much softened and showed light brown discoloration. When sizable pieces of discolored stem tissue were placed in a shallow layer of water, evacuation tubes promptly grew out and within three hours large numbers of motile zoospores were swimming about. Despite a temperature of 29°C. development of zoospores continued more than a day longer. By planting fragments of affected stem on sterile maizemeal agar, mycelia were obtained; these later formed abundant sexual reproductive apparatus and thus made possible the identification of the species.

The same fungus was isolated as causal agent responsible for a wilt that on July 2, 1951, was found affecting some potted bean plants 30 to 35 cm. high, in a greenhouse adjacent to the one in which stem rot had been noted 5 years earlier. In these larger plants the stems were neither much softened nor markedly discolored at the very base, but instead appeared in a somewhat flattened or collapsed condition from the ground level upward for a distance of 5 to 10 cm. No cottony mycelium was visible externally. Aerial development of the fungus had apparently been discouraged by frugal watering as well as by the reduced relative humidity attending the high temperatures -- often in excess of 35°C. -- prevalent in greenhouses during sunshiny summer days. Similar wilting of beans had been observed under glass for several years preceding 1951, the disease each time following the incidence of hot weather in June.

Bean plants affected with cottony stem rot at the Plant Industry Station, either in greenhouses or, during periods of hot wet weather, in field plots, have usually been found attacked by Pythium butleri Subr. Such occurrence of P. butleri is wholly commonplace since this species is wont to appear as an active parasite on many herbaceous host plants virtually every year for one or two weeks during or following periods of hot wet weather. Indeed, sporadic outbreaks of P. butleri take place not only in the middle latitudes of the eastern United States but also in regions considerably farther north. Of about twenty cultures isolated from affected bean pods and cucurbitaceous fruits early in August 1945, when cottony rot developed in spectacular abundance near Beltsville, all were found referable to P. butleri, though the high temperatures and extraordinarily heavy rainfall preceding and accompanying that outbreak should have been equally favorable for P. myriotylum. The bean variety Ranger, according to W. J. Zaumeyer, showed especially severe injury from cottony stem rot during the summer of 1950 in New Jersey, Delaware, and the Eastern Shore of Maryland.

Among the host plants of Pythium species the bean is unusual in permitting continued upward invasion of its stem at relatively advanced stages of growth. Early in August 1951, an irrigated field of beans near Frederica, Delaware, was reported to be seriously damaged through widespread fungus infection extending from the ground line to the lower branches. Diseased specimens from this field amply confirmed the report. Wefts of aerial mycelium in the crotches between the softened watery stem and the similarly affected proximal portions of the lower branches gave an appearance usual in instances of attack by P. butleri and P. myriotylum. Contrary to expectations, however, neither of the two species most frequently offering a cottony display under natural conditions was found responsible for the severe injury. All infected parts that were placed on sterile maizemeal agar gave rise to mycelium of P. ultimum Trow with the promptness usual when that fungus is present in herbaceous tissues as a primary invador. Such extensive invasion of aerial vegetative parts by species of Pythium is assuredly not frequent among crop plants nearly full grown and generally vigorous at the time of infection.

DIVISION OF VEGETABLE CROPS AND DISEASES

THE INCREASED SUSCEPTIBILITY OF THE PALMETTO CUCUMBER TO DOWNY MILDEW IN SOUTH CAROLINA

W. M. Epps and W. C. Barnes

When the downy mildew-resistant cucumber variety, Palmetto, was released to the trade in 1948, it possessed a very high degree of resistance (1). In fact, under field conditions this resistance approached immunity.

The resistance appeared to be due to two distinct factors. First, Palmetto was highly resistant to infection so that inoculation resulted in very few lesions. Second, the lesions which appeared were small and few if any sporangia were produced on them. Consequently, secondary spread occurred so slowly in Palmetto that, even though the disease appeared early in the season, almost no damage resulted. Where Palmetto was interplanted with the susceptible Marketer in experimental plantings, the Marketer served as a constant source of inoculum, and the disease became quite severe in Palmetto (2, 4).

In 1948 and 1949 the acreage of Palmetto was limited bacause of inadequate seed supplies. In both years the authors visited over 90 percent of the commercial fields in the State planted to this variety. No downy mildew was found in any of the fields visited except on one farm where Palmetto was planted adjacent to a field of the Marketer variety. The Marketer was almost a total loss due to downy mildew. On this farm and on the farm of the experiment station, where many susceptible varieties were planted, mildew was present in Palmetto but was not severe. Fungicides, used experimentally to control mildew, resulted in an increased yield of Palmetto only where it was actually interplanted with a susceptible variety (2, 4).

In 1950 and 1951, the picture changed abruptly. All the Palmetto fields visited, even though isolated by several miles from any known susceptible plants, contained downy mildew lesions. Those fields protected with fungicides suffered little, but several unprotected or poorly protected fields suffered severely. Lesions on Palmetto resembled those on a susceptible variety in that they were large and exhibited an abundance of sporangia. The disease spread fairly rapidly in Palmetto; whereas, in the two preceding years it had not even become established. The use of the recommended fungicides in experimental plots in 1950 and 1951 resulted in increases in yields of 48 and 20 percent, respectively, over the unprotected plots.

Ellis (3) noted that the downy mildew fungus sporulated quite profusely on Palmetto cucumbers in Duplin and Pender Counties, North Carolina, in the fall of 1950, and that the crop was rather seriously affected by the disease. He suggested that a new race of the pathogen might be present.

The ability of the pathogen to sporulate so freely on Palmetto foliage was probably the primary factor which enabled the disease to become established and cause severe damage on this variety in 1950 and 1951. Palmetto was still highly resistant to mildew when it was compared with Marketer, but the degree of resistance was much less than it was originally.

Downy mildew was present in South Carolina in all the years from 1948 to 1951 and was so severe on unprotected susceptible varieties in experimental plantings that they were a total loss. It is therefore considered highly improbable that the absence of disease in isolated fields of Palmetto could have been due to the absence of the pathogen in the area. The weather for the four years has varied considerably, but it was sufficiently favorable in all four years for the disease to cause the complete destruction of unprotected or poorly protected susceptible varieties. If it is assumed that the pathogen was present and the weather was favorable for infection in all four years, then the differences observed must have been due to a change in the resistance of the host plant to attack or a change in the virulence of the pathogen.

It is possible, of course, that the increased susceptibility of the Palmetto may be due to genetic changes within the variety. This is highly improbable, since such a change would have had to occur simultaneously in the seed stocks maintained by several different seed companies. There are some differences in the stocks of the various companies, but they are minor when compared to the distinct differences noted herein.

These observations on the sudden and distinct increase in the apparent susceptibility of Palmetto give credence to the idea that a new race of the pathogen is present. Although the source of this race is not known, it could have developed by natural selection within a mixed population of the pathogen.

Prior to 1950 the Palmetto variety was so highly resistant to downy mildew (4) that it was considered possible to grow fall cucumbers without the use of fungicides. Fungicide recommendations for 1950 were directed primarily toward the control of anthracnose, which is sometimes a serious problem in South Carolina in the fall crop. The increased susceptibility of Palmetto, presumably due to a new race of the pathogen, has made necessary a fungicide program as intensive as that

which was used before Palmetto was developed. This fungicide program, which has never given adequate disease control on the susceptible Marketer, has proved satisfactory on the resistant Palmetto.

Literature Cited

1. Barnes, W. C. The performance of Palmetto, a new downy mildew-resistant cucumber variety. Amer. Soc. Hort. Sci. Proc. 51: 437-441. 1948.
2. _____, and W. M. Epps. Some factors related to the expression of resistance of cucumbers to downy mildew. Amer. Soc. Hort. Sci. Proc. 56: 377-380. 1950.
3. Ellis, D. E. Noteworthy diseases of cucurbits in North Carolina in 1949 and 1950. Plant Dis. Reptr. 35: 91-93. 1951.
4. Epps, W. M. The use of fungicides on downy mildew resistant cucumber varieties. (Abstr.) Phytopath. 40: 787. 1950.

SOUTH CAROLINA TRUCK EXPERIMENT STATION, CHARLESTON

VERTICILLIUM WILT OF POTATOES IN CONNECTICUT 1951[1]

W. G. Keyworth[2, 3]

Following reports of premature wilting in many potato fields in Connecticut in recent years a special study was made during 1951 to determine the cause and extent of the complaint. Isolation and reinoculation tests showed that strains of Verticillium albo-atrum pathogenic to potatoes were present in all cases. Since no other wilt producing organisms were associated with any of the outbreaks it may be assumed that they were all caused by the attack of Verticillium. Cases of blackleg were also seen but could readily be distinguished in the field from the Verticillium outbreaks.

Wilting was apparent in scattered fields during late July and early August. Incidence varied greatly at that time from a few widely dispersed plants in each field to (in one case) at least half of the plants. Throughout August the incidence of the disease in already affected fields steadily increased, the stems in some fields being almost entirely dead by the end of the month. The symptoms appeared in other fields also and eventually very few plantings could be found that were completely free of the disease. It is thus apparent that it is widely dispersed in Connecticut potato-growing areas. The most severe outbreaks were in fields where potatoes had been continuously grown for several years. In one such case the grower reported a gradual diminution in crop year by year and estimated that his 1951 crop would be some 30 to 40% below normal. In several fields the disease was prevalent even though no potatoes had been grown during the two preceding years. In one instance scattered wilted plants were found in a field which had previously been cropped with tobacco for ten successive years. No information was available however on the health of the potato seed used for planting.

Symptoms of the disease were identical with those reported for potato wilt in other areas, viz., yellowing and wilting of the leaves from the base of the plant upwards (often in a one-sided fashion) accompanied by a brown discoloration of the vascular tissue of the stem. This discoloration extended into the xylem tissue of the stem end of the tubers. Many tubers with "pink eye" (1, 2) could also be found in affected fields although this symptom was not invariably associated with the disease. Thus in one field where 78 percent of the tubers showed stem end discoloration only 5 percent showed pink eye. In a field inoculation trial of the Green Mountain variety which resulted in fairly severe wilt incidence no pink eye was seen on any tuber.

Fairly severe symptoms were noted on the Katahdin and Green Mountain varieties but the variety most severely affected was Kennebec. This comparatively new variety had been planted fairly extensively on some farms and in at least two cases showed a very high incidence of wilt. Tubers in these fields were severely affected with stem end discoloration and with pink eye. Many also showed a diffused internal discoloration of the flesh which rendered them unsalable.

In a survey to determine the strains of Verticillium found most commonly in Connecticut, potato isolates were made from 23 widely dispersed fields. Eighteen of these yielded isolates which were of the microsclerotial type (V. dahliae Kleb.) and 4 gave isolates of the dark-mycelium type (V. albo-atrum Reinke and Berth). Five intermediate strains were isolated from occasional plants however. They formed both dark mycelium and microsclerotia and could thus not be classified on their cultural characters. Typical isolates from each group were tested for pathogenicity and all were found to be infective. The existence of the intermediate forms lends support to the theory that the dark-mycelium and microsclerotial forms are members of a group species V. albo-atrum and that V. dahliae may not be a valid species. The pure microsclerotial type which was preponderant among the isolates obtained is considered to be sufficiently stable however for it to be readily distinguished from the pure dark-mycelium type. Its widespread occurrence thus appears significant since it has been noted in Connecticut on many other hosts, e.g., eggplant, tomato, maple, etc. It is thus likely that it is a common member of the soil flora in the State and that the main source of infection of the potatoes is the soil in which they are growing. This is further supported by the fact that isolates studied by the author from the Maine seed-growing areas (supplied by Dr. D. Folsom) were all of the dark mycelium type.

---

[1]Report of a study conducted under the Research and Marketing Act of 1946.

[2]Plant Pathologist, East Malling Research Station, Kent, England. Stationed at The Connecticut Agricultural Experiment Station, New Haven, Connecticut, Nov. 1950 - Nov. 1951.

[3]The author is indebted to Dr. A. Hawkins, Potato Specialist, University of Connecticut for permission to collect samples from one of his experiments and for his cooperation in locating disease outbreaks.

The only practical control measures which Connecticut growers can apply at present are (a) the avoidance of very susceptible varieties, (e.g. Kennebec) for planting on ground known to be infested with wilt; (b) the use of wilt free seed for planting new ground; (c) crop rotation to avoid the continuous planting of potatoes. It is considered that a two year planting of non-susceptible crops between successive plantings of potatoes will give some reduction in disease although as already noted this will not rid the soil of the fungus.

An experiment conducted by the University of Connecticut on the reclamation of "run-out" potato ground (3) gave some information this year on fertilizer treatments which might possibly benefit the plants growing in wilt affected soil. High applications of superphosphate or nitrogen resulted in less wilting than on the check plots given balanced fertilizer. High lime dressings on the other hand gave more wilting. These effects would seem to be confined to the appearance of the stems however since an examination of the tubers for stem-end discoloration showed equal incidence of wilt on all plots.

Literature Cited

1. Boyd, O. C. Some facts about Verticillium wilt of potatoes. Univ. of Mass. Extension Circ. (Mimeo.)
2. Folsom, D., O. L. Wyman, and E. Westin. Potato Verticillium Wilt. Maine Extension Circ. 265.
3. Hawkins, A., B. A. Brown, and E. J. Rubins. Extreme case of soil toxicity to potatoes on a formerly productive soil. Amer. Potato Jour. 28: 563-577. 1951.

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

EFFECT OF TUNG-NUT MEAL AND PARATHION ON ROOT-KNOT NEMATODE INFECTION

D. L. Gill

Control of root-knot nematode (Meloidogyne spp.) in the South by the use of tung-nut meal as a fertilizer has been claimed by some nurserymen for several years. Because questions regarding this claim frequently have been asked the writer and no information was available the six tests reported here were conducted. In the last three tests, parathion, as Vapophos, a proprietary material containing 15 percent parathion, was included because parathion had been shown by Dimock and Lear (1) to reduce root-knot nematode infestation.

METHODS

A sandy woods soil was used in all tests. It was placed in No. 10 cans, and the amount of the tung-nut meal or the parathion to be used was calculated on the basis of the surface area of soil in the can. Amounts of tung-nut meal varied from 0 to 5000 pounds per acre. Parathion was used at the rates of 200, 400, and 600 pounds of 15 percent material per acre. The soil for the several cans receiving each treatment was poured together and thoroughly mixed with the meal or parathion. Nematode inoculum was obtained by grinding in a food chopper heavily knotted gardenia roots collected near Spring Hill, Alabama. The quantity of inoculum needed for a test was stirred thoroughly after it was ground, and then it was mixed with the soil at the rate of one tablespoonful per can. Planting was made immediately afterward. The roots of each plant were scored for galling and the relative root-knot index of each treatment calculated according to the method of Smith and Taylor (5).

The tung-nut meal used in tests 1, 2, and 3 was purchased on the open market in December 1949. Subsequently this was considered a possible source of error, since Emmel (3) had shown that the toxicity of tung-nut meal to chicks decreased with the age of the meal. Accordingly, meal milled in May 1950 was compared with the old meal in tests 4, 5, and 6. The new meal was not ground until needed for use.

Emmel (3) also showed that saponin and an unnamed toxic agent are present in tung-nut meal. Saponin was removed by two methods used by him in these tests. In one, one liter of meal was moistened with 600 milliliters of five percent aqueous solution of hydrochloric acid; then, after standing for 30 minutes it was heated for 30 minutes in a pressure cooker at 15 pounds pressure. In the other, the meal was tied in a sack made of six layers of cheesecloth and suspended in running water for 24 hours.

TESTS CONDUCTED

In the first test, 10 cans were used for each treatment; and three tomato seedlings (variety Pritchard) were transplanted to each can December 22, 1949. Plants dying within the first few days were replaced. The plants were grown in a greenhouse that was unheated unless freezing weather was forecast. The plants were removed March 29 and rated as to root-knot index. These ratings, together with the amount of tung-nut meal applied, are shown in Table 1. There was some indication of control at 2500 and 3000 pounds per acre. The plants grown with the highest rate were stunted.

In a second test, 16 cans were used for each treatment and three rooted cuttings of Gardenia jasminoides (variety Veitchi) were transplanted to each can April 13, 1950. These were removed June 14. Root-knot ratings and treatments are shown in the table. No practical value of tung-nut meal was evident. For the third test, these gardenia plants were put back in infested soil containing the same amount of meal as they were grown with previously, because some growers had stated that root-knot infection of transplanted plants becomes less severe in treated soils. These plants were removed November 7 and, as shown in the table, infection was heavy regardless of treatments.

In the fourth test, old and new lots of tung-nut meal, untreated, leached or acid-treated, were compared with parathion. Ten cans were used for each treatment and three tomato plants (variety Pritchard) were transplanted to each can June 9, 1950. These were removed and rated August 3. The ratings (table) show no consistent beneficial effect of the tung-nut meal upon root-knot infection, but parathion reduced it.

Table 1. Relative root-knot nematode index[a] of plants grown in artificially infested sandy woods soil and treated with tung-nut meal or parathion[b].

Treatment (lbs. per acre)	Tomato test 1	Gardenia test 2	Gardenia test 3	Tomato test 4	Squash test 5	Gardenia test 6
Untreated	87	84	100	80	98	53
Tung-nut meal						
Old (1000 lbs.)	76			58	91	47
New (1000 lbs.)				56	93	60
Old (1500 lbs.)	86			91		
New (1500 lbs.)				72		
Old (2000 lbs.)	74	73	94	60	91	50
New (2000 lbs.)				95	94	37
Old (2500 lbs.)	61	73	100	50	92	42
New (2500 lbs.)				75	93	43
Old (3000 lbs.)	35	83	100	96	98	50
New (3000 lbs.)				88	97	50
Old (3500 lbs.)		73	100	70	82	52
New (3500 lbs.)				44	94	37
Old (4000 lbs.)		76	100	100	88	45
New (4000 lbs.)				72	88	40
Old (5000 lbs.)					91	41
New (5000 lbs.)					89	38
Old (2500 lbs., acid-treated)				75	89	40
New (2500 lbs., acid-treated)				83	96	50
Old (3000 lbs., acid-treated)				95	95	50
New (3000 lbs., acid-treated)				63	94	40
Old (2500 lbs., leached)				69		
New (2500 lbs., leached)				95		
Old (3000 lbs., leached)				64	85	29
New (3000 lbs., leached)				85	97	48
Parathion (200 lbs.)				38	25	20
Parathion (400 lbs.)				6	14	8
Parathion (600 lbs.)					12	9

[a]Ratings according to root-knot index system of Smith and Taylor (5).
[b]Vapophos, 15% wettable parathion.

Because of the relatively poor survival of plants in test four, two other similar tests were conducted. In test five, 10 cans were used for each treatment and five yellow crookneck squash seed were planted in each can August 8, 1950. The resulting plants were thinned to three. These were removed and rated September 7. The results (Table 1) show no practical benefit from tung-nut meal, but parathion reduced root-knot infection (Figs. 1 and 2). The mean height of the squash plants grown in soil containing the new meal was significantly less than the mean height of the plants grown in soil with an equal amount of the old meal. This reduction did not occur in plants grown in soil in which the acid or water-treated meal was used, suggesting that saponin was the agent retarding growth.

When the soil was prepared for the fifth test, an equal quantity was mixed for the sixth test. Three rooted cuttings of Gardenia jasminoides received under the variety name Florida were transplanted to each can August 8, 1950. These were removed and rated June 6, 1951. The root-knot ratings (table) show there was not as much root knot present as in other tests, but there was no benefit from the tung-nut meal. Parathion reduced root knot. There was no retardation in growth by the new meal as compared with the old.

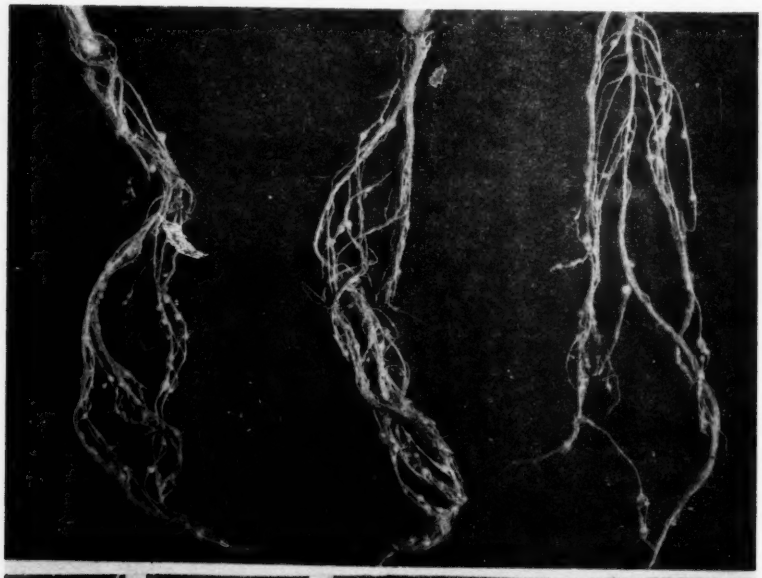


FIGURE 1. Effect of tung-nut meal on root-knot nematodes on squash. Left to right: 1, untreated; 2, 3000 pounds per acre; 3, 5000 pounds per acre.

FIGURE 2. Effect of parathion on root-knot nematodes on squash. Left to right: 1, treated at 600 pounds 15 percent parathion per acre; 2, treated at 200 pounds 15 percent parathion per acre; 3, untreated.

CONCLUSIONS

The results of these tests show that tung-nut meal is of no value as a soil treatment for the control of root-knot nematodes. The benefit some growers have reported is probably due to the fertilizing effect of the meal. Parathion thoroughly mixed in the soil was of considerable value in root-knot nematode control. This had also been demonstrated by Dimock and Lear (1) in pot culture. Ellis and Clayton (2) reported field treatments ineffective. Dimock and Lear (1) stated that this ineffectiveness is probably due to the less thorough mixing in the field. While parathion is of value in pot culture, its practical field use at this stage is questionable. The effect upon other plants is unknown, and it should not be used in soil growing food plants, since Starnes (6)

and Grainger and Leiby (4) reported that it can be absorbed from the soil and translocated to above-ground parts of the plant. Parathion is toxic to humans through skin absorption or otherwise and must be used with great care.

Literature Cited

1. Dimock, A. W. and Bert Lear. Soil treatments with parathion for the control of root-knot nematode and golden nematode. Phytopath. 40: 460-462. 1950.
2. Ellis, D. E. and C. N. Clayton. Soil treatments with new insecticides ineffective in control of root-knot. U. S. Dept. of Agr. Plant Dis. Reptr. 32: 476-477. 1948.
3. Emmel, M. W. The toxic principle of the tung tree. Florida Agr. Expt. Station Tech. Bull. 431: 1-35. 1947.
4. Grainger, M. M. and R. W. Leiby. How plants absorb parathion. Agricultural Chemicals 4 (2): 34-35, 79-81, 83-84. 1949.
5. Smith, A. L. and A. L. Taylor. Field methods for testing for root-knot infestation. Phytopath. 37: 85-93. 1947.
6. Starnes, Ordway. Absorption and translocation of insecticides through the root system of plants. Jour. Econ. Entomology 43: 338-342. 1950

DIVISION OF ORNAMENTAL PLANT CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, TIFTON, GEORGIA

NEW WILDFIRE SYMPTOMS ON SOYBEAN[1]

J. H. Graham[2]

Wildfire of soybean caused by Pseudomonas tabaci (Wolf and Foster) Stapp was first recognized in 1943 (1). Today it is present in most soybean areas of the United States, in some instances reaching epiphytotic proportions.

Symptoms of wildfire have been reported previously only on the foliage of soybean. In 1949 a series of observations and isolations was made to determine if parts of the soybean plant other than leaves were attacked by the wildfire-producing organism. Discolored pods, stems and petioles were collected from plants severely infected with wildfire and pustule bacteria. After surface sterilization the tissues were plated on beef-dextrose and Wernham's potato dextrose agar. Young greenhouse soybean plants were inoculated to test the pathogenicity of the isolates.

The wildfire pathogen was isolated from stems, petioles and discolored tissue on pods and from seed under the discolored areas. Irregularly shaped, brown lesions were very numerous on the stems of susceptible varieties. Brownish spots and streaks were also found on the pulvini, petioles and petiolules. On pods the infected areas were dark brown and somewhat water-soaked with occasional lighter brown necrotic streaks near the margins. Similar water-soaked pod lesions were produced by artificial inoculations on greenhouse plants. In some instances, both the wildfire organism and the bacterial pustule organism, Xanthomonas phaseoli var. sojense (Hedges) Starr and Burk., were isolated from the same lesion on these tissues. Less frequently, only the latter pathogen or no pathogenic bacteria at all were recovered from similar discolorations.

The typical wildfire lesion on soybean leaves consists of a necrotic area surrounded by a large chlorotic zone. Lesions without the chlorotic zone, believed caused by the wildfire organism however, have been found. They were circular light brown necrotic areas, 3 to 8 mm. in diameter with dark brown borders and usually a tiny dark portion in the center. This type of lesion probably develops when the tissue around the infection point is congested with water. Presumably the cells in the area are killed so rapidly by the invading bacteria that no chlorosis results.

Literature Cited

1. Allington, W. B. Wildfire disease of soybean. Phytopath. 35: 857-869. 1945.
2. Valleau, W. D., E. M. Johnson, and S. Diachun. Association of tobacco leafspot bacteria with roots of crop plants. Sci. 96: (N.S.): 164. 1942.
3. Wolf, F. A. Wildfire of tobacco. North Carolina Agr. Expt. Sta. Bul. 246. 26 pp. 1922.

PRESERVATION OF THREE BACTERIAL PATHOGENS OF SOYBEAN IN CULTURE[1]

J. H. Graham[2]

In a previous study involving the three bacterial pathogens of soybean, Xanthomonas phaseoli var. sojense, Pseudomonas glycinea and Pseudomonas tabaci, difficulty was encountered in maintaining actively growing, virulent cultures on common laboratory media. Since the previously reported methods (1, 2, 4) of maintaining virulent cultures of the three organisms for long periods were unsatisfactory, further attempts were made as follows:

In the first test isolates were transferred to beef dextrose agar slants and stored at 4° - 8°C. and tested for pathogenicity after 10, 14, and 26 months by inoculating young soybean plants in the greenhouse. All isolates were alive and virulent after 10 months. After 14 months of storage only P. tabaci and P. glycinea were alive, the former organism still highly pathogenic, the latter only slightly so. After 26 months only P. tabaci was alive and virulent.

---

[1]Cooperative investigations of the North Carolina Agricultural Experiment Station and the Division of Forage Crops & Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture.

[2]Present address, U. S. Regional Pasture Research Laboratory, State College, Pennsylvania.

As a second method, four isolates of each of the organisms were stored in test tubes of beef-peptone broth (no dextrose) at 4° - 8°C. The mouths of one-half of the tubes were covered with tin foil. After 31 months of storage in the tubes covered with tin foil, X. phaseoli var. sojense could not be revived, however, P. tabaci and P. glycinea were viable and highly pathogenic. In the tubes without tin foil caps only P. tabaci was alive after 31 months.

Since mineral oil has been used successfully for the preservation of some phytopathogenic bacteria as well as many other microorganisms for relatively long periods without transfer to new media (3), it was used in a third test with the soybean pathogens. Each of the isolates was inoculated onto two beef dextrose agar slants and allowed to grow one day before it was covered with sterile mineral oil. After storage at room temperature for 29 months all isolates were alive and pathogenic.

Of the three methods tried, storage under mineral oil was the most successful for the three pathogens. This method not only assures the maintenance of virulence for long periods of time but allows for easy transfer from the stock cultures anytime during the storage period.

Literature Cited

1. Coerper, F. M. Bacterial blight of soybean. Jour. Agr. Res. 18: 179-194. 1919.
2. Hedges, F. A study of bacterial pustule of soybean, and a comparison of Bacterium phaseoli sojense Hedges with Bacterium phaseoli E. F. Sm. Jour. Agr. Res. 29: 229-251. 1924.
3. Sherf, A. F. A method for maintaining Phytomonas sepedonica in culture for long periods without transfer. Phytopath. 33: 330-332. 1943.
4. Valleau, W. D., E. M. Johnson, and S. Diachun. Angular leafspot and wildfire of tobacco. Kentucky Agr. Expt. Sta. Bul. 454, 60 pp. 1943.

NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION AND DIVISION OF FORAGE CROPS AND DISEASES

INCREASE OF DUTCH ELM DISEASE IN ILLINOIS

J. C. Carter

Dutch elm disease was first found in Illinois in 1950[1]. One infected tree was found in Coles County. A survey conducted over an area extending from Effingham to Kankakee and east to the Indiana State line failed to reveal any other cases.

During 1951 eleven additional trees affected with the disease were found in four counties (Fig. 1), all near the eastern border of Illinois. These trees were distributed as follows: one in Lawrence County, one in Coles County, five in Douglas County, and four in Champaign County, with a distance of approximately 100 miles separating the southernmost from the northernmost trees.

During late summer, an area extending from Lawrenceville in Lawrence County to Urbana in Champaign County and east to the Indiana State line was scouted systematically by Mr. Ernest D. Bergeson, State Horticultural Inspector. No additional Dutch elm-affected trees were found.

Of the twelve diseased trees so far known in Illinois, five stood in parkways or on city lots, four in highway plantings, and three on farm land but near highways. No explanation for the occurrence or distribution of the diseased trees can be given, but it is noteworthy that none of them have been far removed from railroads or highways carrying heavy traffic from the east.

---

[1] Carter, J. C. Dutch elm disease in Illinois. Plant Dis. Reptr. 35:56. 1951.

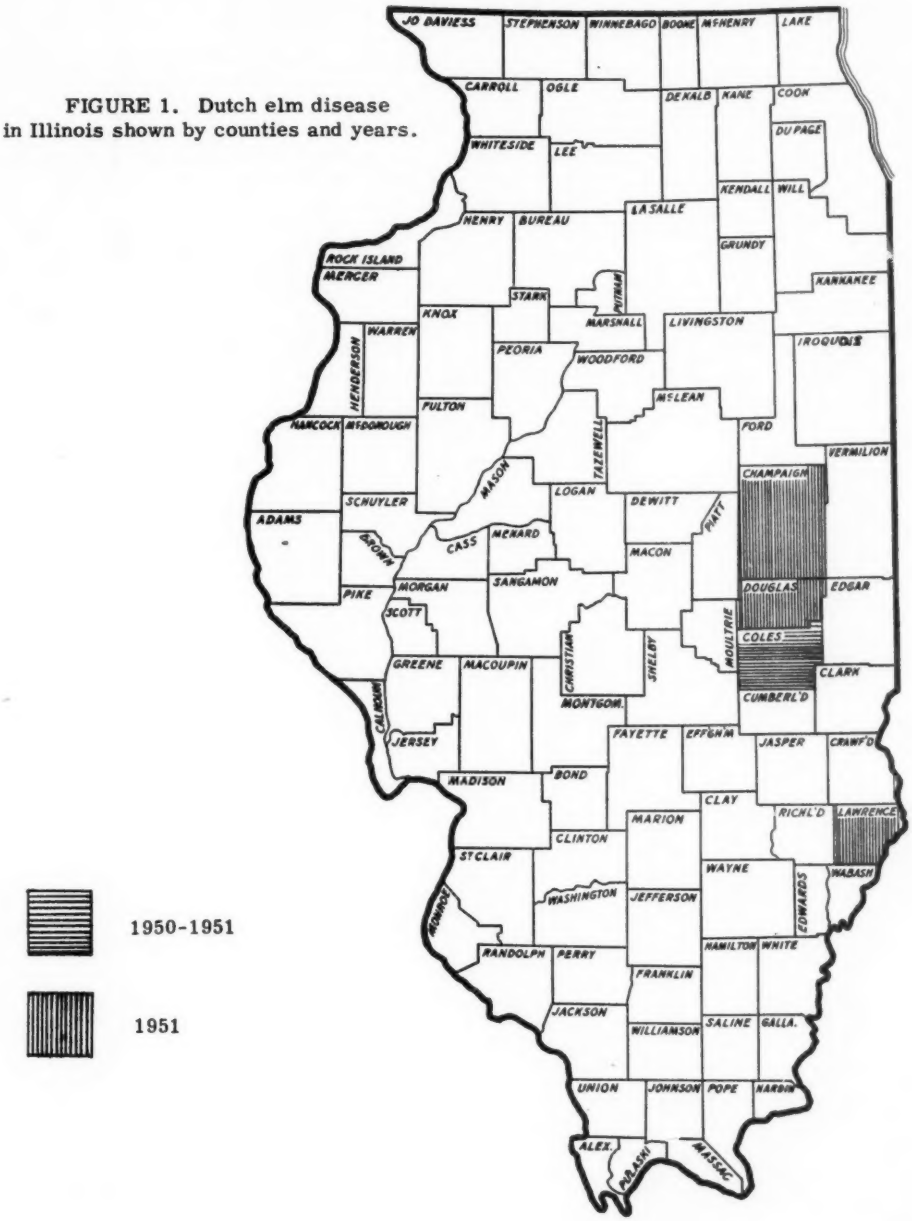


FIGURE 1. Dutch elm disease in Illinois shown by counties and years.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS NATURAL HISTORY SURVEY, URBANA

DISTRIBUTION AND SPREAD OF OAK WILT IN ILLINOIS

J. C. Carter

The known distribution of oak wilt in Illinois in 1947 and spread of the disease since that time are shown on the map in Figure 1. The disease, first proven to be present in the State in 1942[1], was widely distributed in 27 counties in the heavily wooded northwestern section of the State by 1947. Since 1947 the presence of oak wilt has been established in 22 additional counties. This has been determined by laboratory culture tests for 20 counties and by field observations for the other two counties. In these 22 counties, the disease was found in seven (Cook, Will, Kankakee, Iroquois, Randolph, Perry, and Jackson) in 1948, in five (Boone, Du Page, Macoupin, White, and Pope) in 1949, in seven (Lake, Vermilion, Edgar, Coles, Piatt, Wayne, and Calhoun) in 1950, and in three (Sangamon, Madison, and Cumberland) in 1951. As a survey of the State has not been systematically made, oak wilt may be present in some of the unreported counties. However, most of the 53 counties in which the disease has not been found are located mainly in the central part of the State where the land is utilized extensively for growing grain crops and where oak timber is not abundant. In the northwestern part of the State the disease has become seriously destructive in many localities, but in eastern and southern counties it occurs only occasionally.

[1] Carter, J. C. Oak wilt in Illinois. Plant Dis. Reptr. 34(3):81-82. 1950.

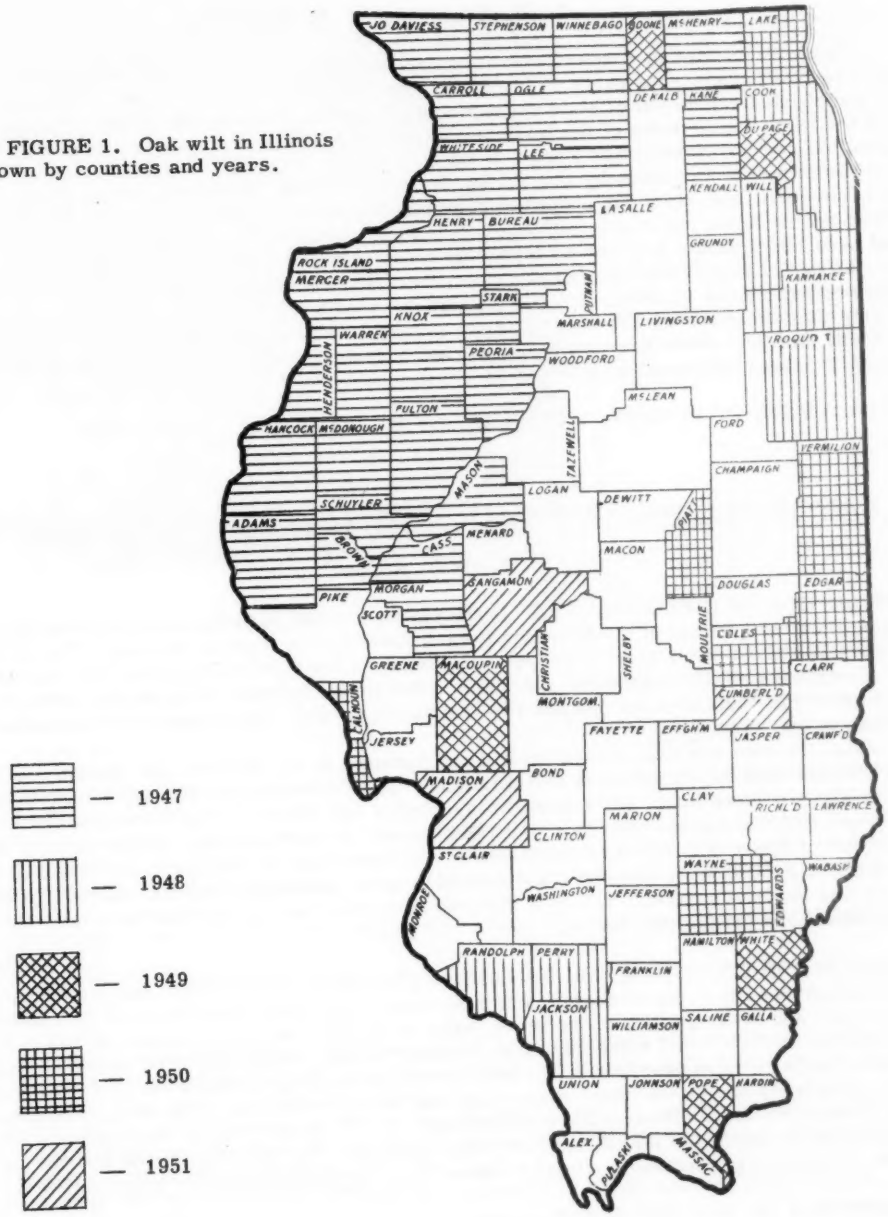


FIGURE 1. Oak wilt in Illinois shown by counties and years.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS NATURAL HISTORY SURVEY, URBANA

OAK WILT IN WESTERN MARYLAND

L. O. Weaver and W. F. Jeffers

Oak wilt, caused by Chalara quercina, was found in Garrett County in Western Maryland on July 19, 1951. A survey party -- including personnel from the Department of Botany, University of Maryland; Department of State Forests and Parks; the Department of Research and Education; and the U. S. Department of Agriculture -- discovered the trees when investigating reports of dying oaks in the mountainous region of the State. The fungus was isolated by Dr. W. Jeffers and confirmation was made by Dr. T. W. Bretz, Columbia, Missouri. Seven trees, including red, chestnut, and white oak appeared to be infected in one location. Some of the trees had been killed two or three years ago, and two red oaks had died in 1951. Only a small number of trees had been killed within four years; there was no evidence of rapid spread of the disease.

An aerial survey with excellent cooperation from the Maryland Wing Civil Air Patrol was made on September 9, 1951. Fifteen aircraft covered a total of nine hundred square miles. Thirty-nine locations of "off-color" trees were located on geological survey maps. Thirty-two of these locations were later visited by ground crews. Isolation attempts from samples of oak failed to reveal additional locations of oak wilt.

DEPARTMENT OF BOTANY, UNIVERSITY OF MARYLAND, COLLEGE PARK

A SPECIES OF GANODERMA PROBABLY PATHOGENIC TO SASSAFRAS IN NEW YORK

Louis G. Nickell[1]

DESCRIPTION OF INFECTED TREE: In the spring of 1950, a large sassafras tree (Sassafras albidum) on the grounds of the Brooklyn Botanic Garden was found to be dead. This tree was between 30 and 40 years old. There were no evidences of injury to the tree, nor had there been any disturbance of soil areas near the tree. A few small fungal fruiting bodies were present on one side of the trunk between one and two feet above ground, which later were identified as a species of the genus Ganoderma.[2]

After the tree was dug up for removal and burning, it was noticed that the entire root system was decayed with the exception of one main root. A section was cut through the trunk about 12 inches above ground level and a large decayed area was found to be present beneath the site of the fungal fruiting bodies. Sections were then made at 6-inch intervals up the trunk to determine the extent of decay (Fig. 1). In this manner it was found that the decayed area extended from over 3 feet above ground level downward and inward at an oblique angle into the sapwood of the trunk and on into the root system. The root that was free from disease was diametrically opposite the area which bore the fruiting bodies.

ISOLATION AND IDENTIFICATION OF FUNGI FROM INFECTED WOOD: Since it was probable that a fungus was the cause of death, isolations were made from various places throughout the root system and lower portion of the trunk, care being taken to remove small pieces of wood under conditions which would allow no outside contamination. These isolations were then placed on various sterile nutrient agar media in order to isolate any fungi in the wood. Of the many isolates made, apparently one or the other of two fungi was always obtained. This was confirmed by Mr. Ross Davidson of the Division of Forest Pathology, U. S. Department of Agriculture, who identified the isolates and found that there were only two fungi: Trichoderma viride and an unidentified species of Ganoderma, probably closely related to G. applanatum and G. sulcatum.

DISTRIBUTION OF THE TWO FUNGI: A check with the description of the site for each isolation showed that when the isolation was made from the fruiting body or any obviously decayed area, the fungus obtained was T. viride. When the isolation was made from the zone of incipient decay, the fungus was always the Ganoderma species. Isolations from beyond the zone of incipient

[1] Research Associate, Brooklyn Botanic Garden, Brooklyn, New York.

[2] It is a pleasure to acknowledge the help of John S. Boyce, Professor of Forest Pathology, Yale University, in the preliminary identification of the fruiting bodies and for his counsel.

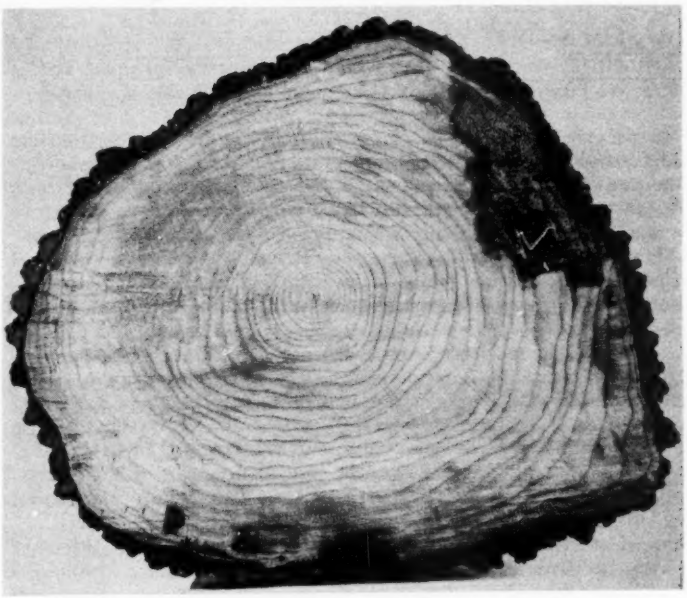


FIGURE 1. Cross-section of sassafras tree trunk made approximately 2 feet above ground level, showing decayed area caused by fungal action.

decay yielded no contaminating organisms.

DISCUSSION OF PATHOGENICITY: Until infectivity tests are run the proof of pathogenicity, of course, cannot be established. Pending such tests, however, we believe that the primary invader and pathogen was the species of Ganoderma and that T. viride was a secondary invader following behind the pathogen, as it has been shown to be in other cases, e.g., after Fomes annosus (1, 2).

The failure to isolate Ganoderma from any of the areas of advanced decay probably is due to the ability of T. viride to overgrow the former on laboratory media. Comparative growth rate studies of these two fungi confirm this and are quite similar to the results obtained by Rishbeth with F. annosus and T. viride (1, 2). The isolation of T. viride from the Ganoderma fruiting body also is not surprising since this fungus has been shown many times to be a parasite in other fungi (3).

Apparently this is the first case of a Ganoderma species having been reported as a pathogen for sassafras.

Literature Cited

1. Rishbeth, J. Observations on the biology of Fomes annosus, with particular reference to East Anglian pine plantations. I. The outbreaks of disease and ecological status of the fungus. Ann. Bot. 14: 365-383. 1950.
2. Rishbeth , J. Observations on the biology of Fomes annosus, with particular reference to East Anglian pine plantations. II. Spore production, stump infection, and saprophytic activity in stumps. Ann. Bot. 15: 1-21. 1951.
3. Weindling, R. Trichoderma lignorum as a parasite of other soil fungi. Phytopath. 22: 837-845. 1932.

BROOKLYN BOTANIC GARDEN, BROOKLYN, NEW YORK

UNUSUAL OCCURRENCES OF PLANT DISEASES

THE REAPPEARANCE OF SILVER LEAF IN THE APPLE ORCHARDS OF WASHINGTON STATE

By Roderick Sprague[1] and H. H. V. Hord[2]

Silver leaf symptoms were observed in 1950 in parts of north central Washington for the first time in a number of years. Heald and Boyle[3] had studied the disease induced by Stereum purpureum in 1923-26 and correlated the activity of the causal organism with winter injury of the trees. The condition was widespread in the Spokane Valley in 1923 following the severe winter of 1919. It had been recognized as causing loss in the upper Columbia River country as early as 1916-17. Therefore, its return in 1950 in all commercial varieties following the two severe winters of 1948-49 and 1949-50 caused some concern. While horticulturists frequently blame all of the situation on the low temperatures, it has been repeatedly demonstrated that Stereum speeds the end of injured trees. In the current outbreak, injury has not been confined entirely to old trees. Some orchards of comparatively young trees have been found seriously diseased. In a few instances in the Methow and upper Wenatchee Valleys young orchards are infected. A five-acre orchard of six-year-old Golden Delicious with 90 percent of the trees silvered was noted in 1950 near Carlton in the upper Methow Valley. It again showed silvering in October 1951 although these trees were not in poor condition and evidently would survive.

In the fall of 1950, scattered trees developed sporophores of S. purpureum; but by the next fall the fruiting bodies were relatively common in the upper portions of the Okanogan, Methow, Entiat and Wenatchee Valleys. In the Wenatchee Valley silver leaf was present from Leavenworth at the upper end to near Cashmere within a dozen miles of the town of Wenatchee. Infection was most prevalent in Okanogan County near Malott and near Tonasket.

There is a campaign in progress to encourage removal of old winter-injured trees that no longer bear a commercial crop. These trees not only bear small crops but the fruit tends to be small, ill-shaped, slow to color and is often off-flavor. Trees with silver leaf symptoms bear such type of fruit in most instances. The removal of these old trees is, of course, aiding in the reduction of silver leaf. One large orchard near Tonasket which was cut down showed that all of the trees were heavily infected with wood-rotting mycelium. The non-invaded wood was confined to a narrow ring adjacent to the bark. The major part of the wood was a discolored ashy-brown typical of the decay noted by Heald and students in 1923-26 in the Spokane Valley.

The chance for recovery of old trees invaded by Stereum is poor. In most cases, they die limb by limb, or sometimes the whole tree succumbs at once. These trees should be removed. Young trees, if given normal care, will continue to be useful, although their life expectancy is somewhat lessened. Adequate fertilizer with moderate irrigation is needed.

In replanting orchards, hardy understock such as Hibernal should be used. Double-worked frameworks of Hibernal, Charlamoff or Antonovka may also prove useful in developing hardy stock resistant to winter and silver leaf. This is the only way that the upper parts of the northern valleys of north central Washington will be able to profitably grow apples in an area where winter injury, silver leaf and perennial canker are constant threats.

WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, INSTITUTE OF AGRICULTURAL SCIENCES, STATE COLLEGE OF WASHINGTON, PULLMAN

[1]Pathologist, Tree Fruit Experiment Station, Wenatchee, Washington

[2]Formerly graduate assistant, Department of Plant Pathology, Washington State College, Pullman.

[3]Heald, F. D. and L. W. Boyle. The Menace of Silver Leaf, Proceedings of Washington State Horticultural Association. 19:50-54, 1923.

PHYTOPHTHORA CINNAMOMI ON AVOCADO IN MEXICO AND COSTA RICA, AND OTHER AVOCADO DISEASES IN MEXICO

By George A. Zentmyer

Phytophthora cinnamomi Rands was isolated from avocado roots collected in Mexico in the course of a trip to some of the native avocado stands and cultivated avocado groves of that country in April, 1951[1]. Root samples were collected from avocado trees in the states of Nayarit on the west coast, and of Puebla, Mexico, Morelos, and Veracruz in eastern Mexico. Only one sample out of thirty yielded P. cinnamomi. This root sample was from a healthy-appearing Mexican-type seedling avocado growing along the road from Coscomatepec to Huastusco in the state of Veracruz. The fungus is evidently native to that region, as the tree from which the sample was taken was growing in a sparsely inhabited, forested area. According to all records examined, this is the first report of this fungus from Mexico.

No evidence of avocado root rot was observed in numerous trees examined in the areas named above, but several other diseases of minor importance were noted. Near Teziutlan in the state of Puebla a leaf spot was observed on Mexican-type seedling trees. Perithecia of Mycosphaerella sp. were present on the spots. The fungus is apparently M. perseae Miles, originally described from Puerto Rico[2]. A similar disease has been described in Florida by Stevens and Piper[3] as Cercospora spot, with which a Mycosphaerella stage is sometimes associated. Another pest of avocado observed near Atlixco, Puebla, was mistletoe (Phoradendron sp.), which had invaded weakened trees to a considerable extent. Leaf spots caused by lichens were noted in several localities.

In September 1951, Phytophthora cinnamomi was isolated from avocado roots sent to Riverside, California, under quarantine permit from Costa Rica. According to a description of the disease in a letter from E. J. Fleener of the Institute of Inter-American Affairs, San Jose, Costa Rica, the trees showed typical symptoms of root rot. Considerable losses are resulting from the disease in the avocado-producing regions of Costa Rica.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA CITRUS EXPERIMENT STATION, RIVERSIDE

[1]The writer expresses deep appreciation to Dr. J. G. Harrar, Field Director for Agriculture, The Rockefeller Foundation Laboratory, Office of Special Studies, Mexico City, and to Dr. J. S. Niederhauser, Pathologist in the same laboratory, for extending use of laboratory facilities and for aid during the stay in Mexico.

[2]Miles, L. E. Some new Porto Rican fungi. Illinois State Academy of Science Trans. 10:249-258. 1917.

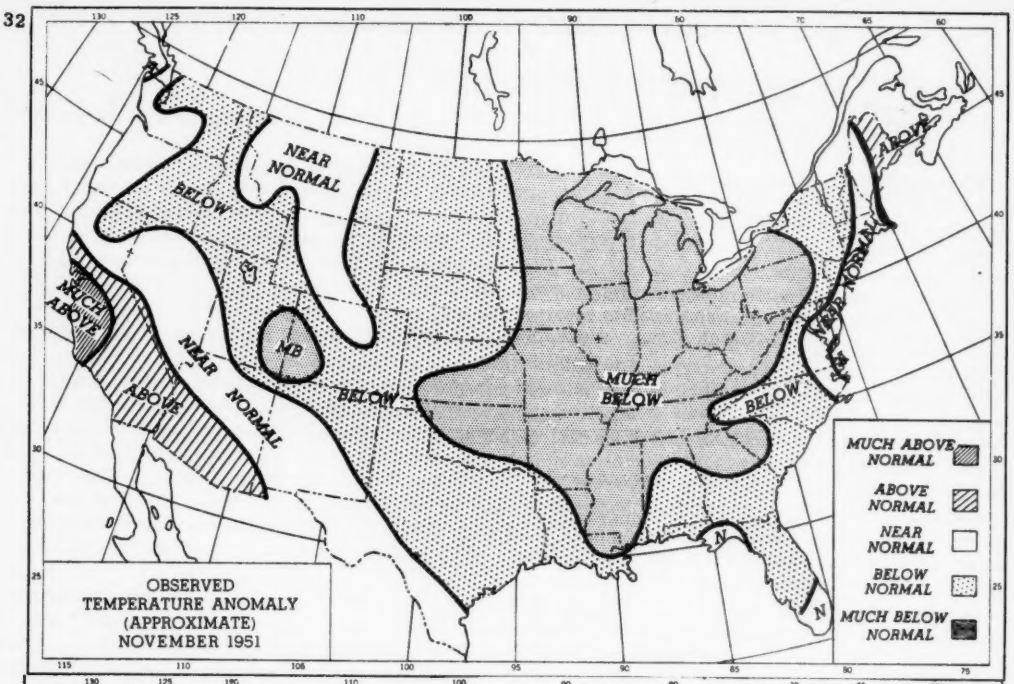
[3]Stevens, H. E., and R. B. Piper. Avocado Diseases in Florida. U. S. Dept. Agr. Cir. No. 582. 1941.

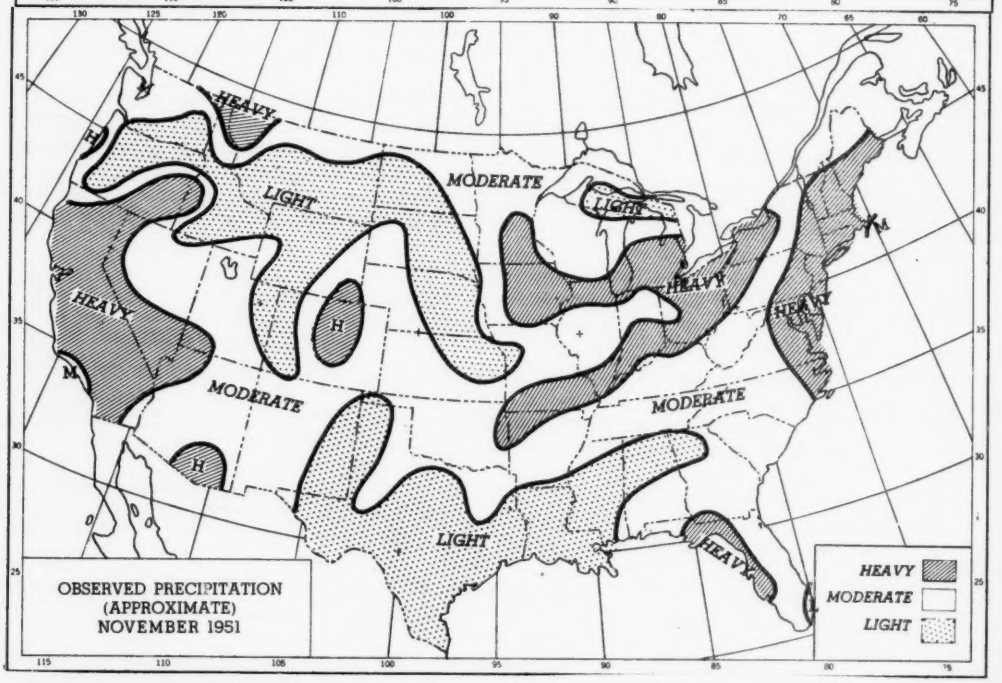
SOME CORRECTIONS

Robert H. Fulton sends the following footnotes to be included in two papers in the December 15, 1951 number of the Reporter:

1. Comparison of Fungicides for Control of Powdery mildew on the Latham Red Raspberry in 1950, by Robert H. Fulton, page 538.
   Footnote:
   Published with the approval of the Director as Journal Article 1302 of the Michigan Agricultural Experiment Station. Contribution 51-10 Department of Botany and Plant Pathology, Michigan State College.

2. A Field Test of Several Fungicides for the Control of Powdery Mildew on Lucretia Dewberry in 1951, by William Young and Robert H. Fulton, page 540.
   Footnote:
   Published with the approval of the Director as Journal Article 1301 of the Michigan Agricultural Experiment Station. Contribution 51-11 Department of Botany and Plant Pathology, Michigan State College.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.